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INFORMATION REPORT

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SUBJECT	Diseases of Maize/Bibliography
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/Available on loan in CIA library is a Study on the Disease of Maize, prepared by a privately-endowed US research institute./

I The treatment of the diseases of maize as set forth in the study, is somewhat incomplete, has one inaccuracy, omits some information of recent date, and over or understates some pertinent items. Some of these are the following /the following numbered items, 1-16, inclusive, correspond with the numbers appearing in the above-mentioned study./

1. Zea mays includes not only varieties (pod, flour, flint, pop, sweet and dent) but numerous races (4, 22).

2. Reference to 100% losses over-states losses from smut.

3. In comparison with open-pollinated corn, modern corn hybrids are relatively free of smut; probably because (a) susceptible inbred lines and hybrids have been discriminated against in making-up commercial hybrids, (b) the susceptibility of any one inbred line is diluted by the two or more additional inbreds that go into the make-up of a commercial hybrid, and (c) the rapidity of growth and maturation of hybrids seems to suppress smut expression. Barreness from smut infection is uncommon in hybrid corn and, therefore, reference to Garger's and Hoover's observations (which are based on susceptible

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inbreds) over-states the importance of this condition. In fact, some of the greater expression of smut on barren plants may be the result, rather than the cause of barrenness (See Davis'work). Davis noted that more plants in a field were infected than could be detected from visible galls because such plants had rudimentary galls at axillary buds along the stalk, which could be detected only when the leaf sheaths were stripped back.

4. I am inclined to accept Davis' conclusions that most smut infections of corn are of a systemic rather than of a local nature. I base this belief on my observation of the abundance of rudimentary galls at the lower nodes of plants.

5. Because corn smut is a minor disease in the corn belt of the US, farmers do not consciously sanitate against it, nor do the state extension services publicize the need for sanitation.

6. As indicated above, some progress has been made in the US toward developing smut resistant corn.

7. Helminthosporium turcicum also occurs on teosinte.

8. Severe in Connecticut in 1889 (15), in Long Island, New York, in 1897 (14), and in Connecticut, Delaware, eastern Pennsylvania and New Jersey in 1903 (9).

9. Severe in southeast Iowa in 1950 (20), and in the southern and central parts of Iowa in 1951 (12).

10. Symptoms could be described better (10, 17).

11. The species that should be named here is H. carbonum (16, 17, 19), not H.-maydis. Helminthosporium carbonum (Races 1 and 2) is of minor consequence on corn thus far in the US, occurring chiefly on a few susceptible inbreds (10, 16, 17). Helminthosporium maydis is of greater consequence (10, 17).

12. There is more information on resistance in corn to H. turcicum (2, 7, 8, 17, 23).

13. Rust severity in Iowa in 1950 (20, 21) and 1951 (12).

14. Also from Venezuela (13).

15. There was a local epiphytotic of Puccinia polysori in Guatemala in 1951 (5).

16. I feel this section should be treated in detail as the other sections have been treated. Some recent pertinent references (1, 3, 11, 12, 18) are included in the attached bibliography.

II. In part from personal experience, I feel that, with proper weather conditions, Helminthosporium turcicum, H. maydis, Puccina Sorghi, P. polysori, Sclerospora phillipinensis, S. maydis, S. macrospora, possibly other Sclerospora spp., Physoderma Zea-maydis, Cercospora zeae, and C. zea-maydis possess ephiphytotic potentials on corn that are equivalent to the rust fungi on small-grained cereals. Sphacelotheca sorghi, Bacterium stewartii, Phytomonas syringae, Pseudomonas albopræcipitans, Angiospora zeae and viruses possess less epiphytotic potential because (a) they do not reproduce and become disseminated so rapidly, (b) they deteriorate under humid conditions (Sphacelotheca sorghi), or (c) they require locally adaptable insects for transmission. Ustilago maydis and other unnamed pathogens have low

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epiphytotic potentials in my judgement.

III. The absence of consistently large scale epiphytotics of corn diseases in central and South America is due in part to the wide genetic heterogeniety of the corn in those areas and in part to temperature and moisture conditions that vary with the physiography of the regions. In the US, where present corn hybrids are built from inbreds derived from uniform stock, the absence of widespread epiphytotics is due less to genetic heterogenicity but more to low quantities of inoculum coincident with unfavorable weather in critical areas. The low quantities of inoculum doubtlessly are due (a) to corn being a one season crop throughout the US, being planted about the same time of the year at various locations, so that opportunity is not afforded the pathogens to build up on a winter corn crop in the south for subsequent transport to the north, as is the case with rust diseases of cereals, (b) to the inability of spores being carried long distances because either they are too dense or subject to rapid inactivation by dessication, and (c) to the north-easternly direction of the prevailing summer winds, which would tend to keep pathogens confined to the south easternly part of the US, and away from the heart of the cornbelt. Cornbelt corn probably would sustain severe epiphytotics if weather conditions were favorable for mild epiphytotics in the south central states, and from where spores could be wind-transported northward to it. Weather conditions in the cornbelt until Aug usually are favorable for epiphytotics, but because the inoculum is not present, such epiphytotics do not develop. Helminthosporium turcicum epiphytotics have been produced artificially in the field in central Iowa by spraying spores onto the plants, but in one year the fungus failed to sporulate thereon.

IV. The list of pathogens possessing epiphytotic potential of the first order have already been enumerated under (I) above. I think all of them should be considered.

V. Recent available publications to my knowledge have already been indicated.

VI. Additional US Specialists in the pathology of maize are the following: (a) I. E. Melhus, Iowa State College; (b) A. J. Ullstrup, Purdue University; (c) J. G. Dickson, University of Wisconsin; (d) J. J. Christensen, University of Minnesota; (e) C. C. Wernham, Pennsylvania State College.

VII. Bibliography

1. Abbott, E. V., and C. A. Schexnayder. A sclerospora infection of sugar cane in Louisiana. Phytopath. 43:289. 1953. (Abst).

2. Elliott, C., and M. T. Jenkins. Helminthosporium turcicum leafblight of corn. Phytopath. 36:660-666. 1946.

3. Farrar, L. L. Johnson grass as a possible wild host of sclerospora disease of sugar cane. Plant Dis. Reporter 37:446. 1953.

4. Melhus, I. E. Plant research in the tropics. Iowa Agr. Exp. Stat. Res. Bul. 371. 1949.

5. Melhus, I. E. A preliminary study of the diseases of corn and some related hosts in Guatemala. Iowa State College Jour. Sc. 27:519-536. 1953

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6. Melhus, I. E., and R. I. Jackson. Corn growing in Indonesia and some suggestions for increasing production. Landbouw 24: Nos. 7-12. 1952.

7. Melhus, I. E., G. Semeniuk, and E. F. Vestal. In Plant research in tropics. Iowa Agr. Exp. Sta. Res. Bul. 371. 1949. pp. 615-624.

8. Melhus, I. E., and F. O. Smith. Helminthosporium leaf blight of corn in Guatemala. Turrialba 3:14-22. 1953.

9. Orton, W. A. Plant diseases in 1903. Yearbook U.S.D.A. 1903:550-555. 1904

10. Roberts, Alice L. Some of the leaf blights of corn. Yearbook U.S.D.A. 1953:380-385.

11. Semeniuk, G., I. E. Melhus, J. R. Wallin, G. L. Gilly, and Muriel O'Brien. A dwarfing and witches Broom of corn in Iowa. Phytopath. 36:410. 1946. (Abstr.).

12. Semeniuk, G., and E. F. Vestal. Leaf diseases of corn in Iowa in 1951. Plant dis. Reporter 36:171-7,1952.

13. Standen, J. H. Host index of plant pathogens of Venezuela. Plant Dis. Reporter Suppl. 212. 1952.

14. Stewart, F. C. Report of the mycologist. N. Y. Agr. Exp. Sta. 15th Ann. Rept. 1896:449-521. 1897.

15. Thaxter, R. Report of the mycologist. Conn. Agr. Exp. Sta. Ann. Rept. 1889:127-202. 1890.

16. Ullstrup, A. J. Further studies on a species of Helminthosporium parasitizing corn. Phytopath. 34:214-222. 1944.

17. Ullstrup, A. J. Leaf blights of corn. Purdue Univ. Agr. Exp. Sta., Sta. Bul. 572. 1952

18. Ullstrup, A. J. Observations of crazy top of corn. Phytopath. 42:675-680. 1952.

19. Ullstrup, A. J. and A. M. Brunson. Linkage relationships of a gene in corn determing susceptibility to a Helminthosporium leaf spot. Jour. Amer. Soc. Agron. 39:606-609. 1947.

20. Vestal, E. F., and G. Semeniuk. Puccinia sorghi and Helminthosporium turcicum on corn in Iowa. Plant Dis. Reporter 35:213-214. 1951

21. Wallin, J. R. An epiphytotic of corn rust in the north central region of the US. Plant Dis. Reporter 35:207-211. 1951.

22. Wellhausen, E. J., L. M. Roberts, and E. Hernandez X. in collaboration with Paul C. Mangelsdorf. Races of maize in Mexico. Their origin, characteristics and distribution. The Bussey Institution of Harvard Univ. 1952.

23. Wernham, C. C. The mass production of blight resistant corn. Plant Dis. Reporter 37:138-141. 1953.

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MAIZE

In terms of total world production, maize is the fifth-ranking agricultural crop, surpassed by sugarcane, potatoes, rice, and wheat. In total tonnage traded, however, maize ranks in third place, behind only wheat and sugar. In terms of total do value of agricultural exports, maize was in eighth position, slightly below coffee, in the prewar period. Maize thus holds an important place among agricultural crops in world production and trade.

Two countries — Argentina and China — stand out in the economy of maize. In recent years, Argentina has consistently been the most important maize exporting country in the world, with United States maize export tonnage exceeding that of Argentina only occasionally. In 1948, for example, Argentina exported slightly more than 2.5 million metric tons of maize, one-half of total world exports, compared with United States' exports of 600,000 metric tons. No other country even approached Argentina in this respect.

With the exception of the United States, whose maize production is greater than that of the rest of the world combined, China is the leading maize producer. Unlike Argentina, where the bulk of the crop is exported to foreign countries, China uses its maize for domestic purposes and exports little, if any. Brazil is the world's third-ranking producer of maize, with the possible exception of Rumania, where accurate production figures are unavailable.

In Argentina, maize occupied more acres than any other crop except wheat, during the prewar years. Alfalfa acreage exceeded that of maize in 1947, but the prewar relationships may be restored under Argentina's new agricultural program. In 1938, maize was Argentina's fourth most important export commodity, accounting for 12.9% of the dollar value of all exports, and outranked only by meat, wheat, and linseed. By 1944, however, maize represented only 1.8% of Argentine exports. In China, rice, wheat, rapeseed, and barley occupied a greater total acreage than did maize in 1941.

1

Maize is a warm-climate annual plant represented by the single species Zea mays L. The species consists of several varieties, namely; pod, flour, flint, pop, sweet, and dent. Maize is a very adaptable plant and is grown under a wide range of environmental conditions. The native home of maize is in either Central or South America but no wild or uncultivated species are known. Cultivated maize is attacked by numerous pathogens, most of which do not result in marked yield reductions. Ustilago maydis (DC) Cda., Helminthosporium spp., Puccinia sorghi Schw. and Sclerospora will be considered.

Maize Smut

carbon del maiz, or bolsas del maiz. The pathogen is commonly designated *Ustilago maydis* (DC) Cda., although the name *U. zeae* (Beckm.) Ung. is also widely recognized.

Ustilago maydis attacks only *Z. mays* and *Euchlaena mexicana*. The smut galls appear on any part of the host plant where embryonic tissues are exposed. Galls developed on maize seedlings may result in the death of the plants but later gall development rarely kills the plant or plant parts. The galls may develop to a diameter of four to six inches. At first the galls are covered with a greenish white, firm, glistening membrane. As the gall matures, the membrane dries and weathers away, releasing the innumerable chlamydospores of the fungus.

(2) The average annual losses due to corn smut are usually estimated at three to five percent although losses in individual fields may sometimes be nearly 100%. Several investigators have studied the variations in yield losses due to *U. maydis*. (3) Garber and Hoover (1928) concluded that the greatest losses from the pathogen came about as a result of induced barrenness. They found that 1188 smut-free plants were 20% barren, whereas of 868 smutted plants, some 38% were barren. Immer and Christensen (1928, 1931) found that the larger the galls on the stalks, the greater the reduction in harvested grain; also galls above the ears were associated with greater yield reductions than those below the ears. Johnson and Christensen (1935) concluded that the average reduction in yield per plant due to infection by *U. maydis* was approximately 30%. On the average, single smut galls reduced yields about 25% and multiple galls approximately 50%. Single or multiple smut galls above the ears were about twice as destructive as galls located below the ears. Davis (1936) reported the results of observations in maize fields near Ames, Iowa. Eleven percent of the plants in 1930 had visible smut galls, 9% in 1931, 14% in 1932, 6% in 1933, and 18% in 1934. Nemlienko (1941) reported percentages of smut infection in the Ukraine, ranging from less than one to more than 20%. Pepper and Haenseler (1944) reported that annual losses of 5% or more were to be expected in the sweet corn crop in New Jersey, and that losses sometimes were as much as 60% on highly susceptible hybrids.

The smut galls are, in reality, large masses of reddish-brown to black chlamydospores which are finely echinulate, spherical to slightly irregular in shape, and 9 to 12 µ in diameter (Dickson - 1947). The chlamydospores germinate by producing promycelia which usually bear four lateral basidiospores or sporidia. The basidiospores may multiply by budding to produce secondary basidiospores. The sporidia germinate by producing germ tubes which penetrate the epidermis of young cells directly. Walter (1934) concluded that stomata were relatively unimportant as ports of entry; also that chlamydospores sometimes produced germ tubes which penetrated young tissues. Walter believed that chlamydospores were important in initiating secondary infections. Appressoria were not formed. Walter also observed that germ tubes of the pathogen penetrated and affected tissues that were apparently too mature to produce galls. Meristematic activity and hypertrophy of the host cells seemed to be necessary for the formation of the gall.

Many studies of U. maydis have been made on culture media in the laboratory (Christensen and Stakman - 1926, Stakman and Christensen - 1927, Kernkamp and Martin - 1941, Kernkamp - 1942, Stakman, et al. - 1943, 1948, et.al.). It appears that the fungus comprises an indefinite number of biotypes that differ either widely or slightly in every observable character or combinations of characters. At least 5000 easily distinguishable biotypes were obtained from two haploid basidiospores of opposite sex. New biotypes are constantly being producted as a result of mutation and of recombinations resulting from interbiotypic hybridization. Some lines were found to be extremely mutable and others were very constant. Some lines were strictly mycelial in habit, others formed only basidiospores, whereas still others were a combination of the two types. There are multiple sex groups but there is relatively free interbreeding among the biotypes. Notwithstanding the extreme variations encountered in culture, the chlamydospores were observed to be surpisingly uniform in morphology. Mycelium from basidiospores of opposing sex groups must be present if the pathogen is to develop actively in the host tissues, although some few biotypes proved capable of inducing the formation of galls when inoculated signly into maize plants. Stakman, et al. (1948) found that the addition of uranium nitrate to the culture medium stimulated mutation in U. maydis.

The basidiospores and chlamydospores of U. maydis are wind blown from plant to plant and from region to region. The chlamydospores are also quite resistant to unfavorable climatic conditions, gastric juices of animals, etc.

Soil-borne inoculum is the most important source of natural infection of maize plants. According to Chester (1947) chlamydospore germination in the soil is favored by an acid soil reaction. Although chlamydospores may be carred on maize seed, this is not believed to be a major source of inoculum. When young seedlings are infected the mycelium may become systemic, but most infections appear to be local in character. Davis (1936) concluded that most, if not all, smut infection was systemic in character.

Melhus, et al. (1941) found that under controlled environmental conditions the extent of gall formation by U. maydis was increased by moderate wilting of the maize plants, and was decreased by the presence of water in the leaf whorl. [illegible] showed that in the first two years, when the precipitation was greater by 100 mm and the air and soil humidities higher by 16 and 5%, respectively, than in the following two years, the percentage of maize smut was much lower (8.4 to 8.8% as against 17.7 to 20.2%). Greenhouse experiments also showed low moisture correlated with a higher incidence of smut. Maize smut was found to be much greater in the dry Dnepropetrovsk region (9.3 - 16.1%) than in the more humid Rostov (0.3 - 0.7%) and Moldavian (0.3 - 3.5%) regions. Nemlienko believed that brief summer showers and dews sufficed for germination of the spores at _U. maydis_. Chester (1947) wrote that the pathogen was adapted to warm weather, the optimum temperature for spore germaination being 27° to 33°C.

5 Sanitation and crop rotation (three years or longer) are frequently recommended for alleviating the incidence of maize smut. The intensive cultivation of maize in many regions, and the fact that the fungus spores are air borne for considerable distances lessens the value of these measures. Likewise, seed treatment, although sometimes recommended, is ineffective.

Pepper and Haenseler (1944) reported that it was possible to control both the European corn borer and maize smut in sweet corn in the same operation by using dusts containing rotenone or nicotine as the active agents. Generally speaking, control of maize smut with either sprays or dusts has been considered impractical.

Garber and Hoover (1928), Immer and Christensen (1931), Stringfield and Bowman (1942), et al. have indicated that considerable differences in susceptibility to _U. maydis_ exist in _Z. mays_ so that control by the development of resistant varieties is a possibility. Pepper and Haenseler (1944) indicated that sweet corn varieties with tight husks and long husk tips were less subject to smut than loose-husted, short-tipped

6 varieties. Breeding corn for smut resistance has not progressed far in any country.

Helminthosporium Blights of Maize.

The diseases caused by various species of _Helminthosporium_ on _Z. mays_ are commonly designated leaf blight, _Helminthosporium_ blight, and tizon de la hoja del maiz. Several species of _Helminthosporium_ have been reported as attacking maize in different parts of the world. The most prevalent species are _H. turcicum_ Pass., _H. maydis_ Nisikado and Miyake (_Cochliobolus heterostrophus_ Drechs.), and _H. carbonum_ Ullstrup.

7 _Helminthosporium turcicum_ is distributed widely over the world on maize, and various species of _Sorghum_; _H. maydis_ also is widely distributed on maize and teosinte. _Helminthosporium carbonum_ has been reported thus far only on maize in the United States. These species of _Helminthosporium_ are primarily leaf parasites but _H. carbonum_ also attacks the ears of maize.

The leaf spotting conditions caused by the various species of Helminthosporium have been considered as unimportant diseases of maize until recent years, when considerable destruction occurred on certain inbred lines and hybrids of maize.

8

Helminthosporium turcicum was reported as a common and destructive pathogen of maize in Argentina by Campi (1939) and Marchionato (1948). The disease is especially destructive in warm, wet summer and fall seasons. Walker and Magruder (1943) reported that 1942 was an epidemic year for Helminthosporium blight (presumably H. turcicum) in Maryland; open pollinated varieties of maize were damaged less than hybrids. Helminthosporium turcicum was reported by Ellett (1943) as present in Ohio from 1939 to 1943; it was serious on many commercial hybrids. Wernham (1946) reported H. turcicum and H. carbonum as present on maize in Pennsylvania, and H. maydis as epidemic in Chester County in 1945. Helminthosporium turcicum was observed (Hoppe - 1953) to be causing premature drying in maize fields in south-central Wisconsin in 1952. The diseased fields were conspicuous from the highway. Considerable damage occurred since the ears were in the milk stage. The disease was common elsewhere in Wisconsin and had been present in previous years, but was not so widely distributed nor so severe as in 1952. No estimates of the destructiveness of H. carbonum were made by Ullstrup (1944)

9

The leaf lesions caused by H. turcicum appear as long elliptical areas, greenish brown in color, and show concentric zones of different shades of brown. Heavily infected leaves appear as if fired, and very little green area remains (Ullstrup - 1943). The lesions bear conidiophores on both surfaces of the leaf. The olivaceous to brown conidiophores usually emerge through stomata and bear fusoid to ellipsoid-shaped conidia which measure 80 to 200 x 5 to 12 μ in size. The conidia are five to eight septate and have pointed ends. The fungus grows readily in culture (Marchionatto 1948).

The lesions caused by H. maydis are quite distinct from the lesions of H. turcicum The spots are elongate, buff colored with a reddish-brown margin, and have a definite zonate or targetlike pattern. Severe infection may reduce the leaf area sufficiently to affect markedly the yields. Conidiophores, which are olivaceous in color, emerge fr the stomata and are 120 to 170 in length. The conidia are somewhat curved, light olivaceous in color, and are 30 to 115 x 10 to 17 μ in size. Perithecia of Cochliobolus are quite numerous in old lesions. The asci are numerous, 160 to 180 in length, and usually contain filamentous ascospores (Dickson- 1947). Helminthosporium maydis is most prevalent in the southeastern part of the United States.

10

The leaf lesions caused by H. carbonum appear as narrow irregular, chocolate-brown spots. There are at least two races of H. carbonum and both attack the ears, [illegible] the kernels giving the ear the appearance of being charred. The physiologic races are separable on the basis of symptoms produced and the specialization evidenced in parasitism. The conidiosphores, which arise singly or in small groups from stomatat, are olivaceous brown in color, 90 to 230 x 5 to 7μ in size and bear one to several conidia. The conidia are 25 to 100 x 7 to 18μ in size (mean 63 x 13μ), widest in the center and taper toward rounded ends. They are dark olivaceous brown, straight or slightly curved, and two to twelve septate (Ullstrup - 1944).

The principal sources of inoculum of the species of Helminthosporium are the conidia produced on the diseased tissues. Conidia are produced in abundance, are widely disseminated by wind currents, and germinate by producing one or more germ tubes which penetrate the host tissues directly or through stomata (Ullstrup - 1943, Marchionato - 1948).

According to Campi (1939), H. turcicum was described on maize in 1876 by Passerini. The pathogen was mentioned for the first time in France in 1903, in India in 1907, in South Africa in 1912, and in Australia in 1915. Leaf blight is an important disease of maize in middle Europe, Africa, Australia, Russia, India, the Philippines, etc. The fungus is common and often destructive in Argentina. Campi observed that the degree of infection, as well as conidial production, was directly related to the relative humidity. The fungus develops well on ordinary culture media, growing more rapidly at 28°C. than at 20°C., but it produces conidia more abundantly and of larger size at 20°C. Conidia produced in culture are generally smaller than those produced on leaves. The fungus grow best on media at a pH of 5.7.

The species of Helminthosporium attacking maize are said to survive from one season to the next on old infected leaves in the field.

Marchionatto (1948) advised that H. turcicum was best combated by burning the old diseased leaves, rotating maize with nonsusceptible plants, and planting resistant varieties. Dickson (1947) recommended similar measures.

Ullstrup (1941a) pointed out that the dent corn inbred line Pr was unique in its susceptibility to Race 1 of H. maydis; all other inbred lines and hybrids were resistant. The susceptibility of the inbred line Pr was inherited as a monogenic recessive. Ullstrup (1941b) also desribed two physiologic races of H. maydis in the corn belt. Race 1 was proposed to designate the race to which the inbred line Pr is susceptible. Race 1 may have been indigenous to the corn belt or it may have arisen as a mutant from the less specialized type known to occur in Florida. Race 2 was designate to include the race observed on the inbred line 187-2 and a number of others; Race 2 was though to be indigenous to the corn belt but had escaped recognition until the proper environmental condtions ensued and a relatively susceptible hos variety of hybrid was present.

Ellett (1943) noted differences in susceptibility to H. turcicum in Ohio among various commercial hybrids. He was unable to detect any seedling resistance in greenhouse experiments among 24 inbred lines, 26 single crosses, and six- double
12 cross hybrids. Wernham (1946) in Pennsylvania reported that in a greenhouse inoculation experiment, two inbred lines, which were very susceptible to H. turcicum, showed pronounced resistance to H. maydis

Maize Rust

The disease is known commonly as rust. The pathogen is designated Puccinia sorghi Schw.; however, at least two other rust fungi, P. polysora Underw. and Angiopsora zeae Mains, are recognized on maize.

Puccinia sorghi attacks the leaves of Zea mays and Euchlaena mexicana (teosinte). The alternate hosts of P. sorhi are various species of Oxalis, O. Corniculata, O. cymosa, O. stricta, and O. violacea. Puccinia polysora and A. zeae attack the leaves of maize plants; no other host plants have been reported.

The rust diseases of maize have not received nearly so much attention as the rusts of wheat, oats, barley, etc., because they have never been considered of much importance. In most years P. sorghi occurs on maize so late in the growing season that practically no losses can be attributed to the pathogen. However, given favorable environmental conditions early in the growing season, the rust fungus can and does
13 cause considerable reduction in yields of maize. Zogg and Salzmann (1947) reported that destructive attacks of P. sorghi on maize occurred in the years 1942 through 1946 in the low Rhine Valley of St. Gall. The plants were killed in July and August before the ears had ripened. Spraying for control proved ineffectual, so that attention was directed to exterminating the alternate host, O. stricta, in the region.

An anonymous note (Anon. - 1951) in World Crops indicated that there was a destructive rust epidemic of maize in West Africa in 1950. The Government of the Gold Coast found it necessary to import more than 12,000 tons of grain to meet the food shortage occasioned by the 1950 epidemic which swept the maize-growing regions from Nigeria to the Ivory Coast. Rhind, et al. (1952) reported that a virulent epidemic of maize rust began in Sierra Leone in 1949, and was estimated as causing up to 50 percent loss of the maize crops in Liberia, Ivory Coast, Gold Coast, Dahomey, and Nigeria in 1950. Prior to 1949, only the uredospore and teliospore stages of P. sorghi has been collected in West Africa on maize. Due to the virulence of the 1949-50 outbreaks and also to the larger size of the uredospores, Bisby of the Commonwealth Mycological Institute concluded that the rust fungus involved in the maize epidemic was P. polysora rather than P. sorghi. The 1949 collection from Sierra Leone was said to be the first collection of P. polysora outside the Western

Hemisphere. Nattrass (1953) reported severe outbreaks of rust caused by P. polysora in Kenya in June, 1952. Both P. sorghi and P. polysora occur in Zanzibar, Uganda, and Tanganyika, often on the same maize leaf. Nattrass indicated that all the records of severe attacks of P. polysora have been from the warmer, more humid tropical areas.

According to Cummins (1941), there has been considerable confusion in the identification of the rust pathogens on maize. Cummins first discovered that a specimen labeled P. sorghi from Peru was actually P. polysora. Subsequent examinations in the Arthur herbarium showed that the mistake had been made several times in the past, and that P. polysora, formerly believed to occur only on species of Erianthus and Tripsacum, actually occurred as a parasite of maize in Peru, British Honduras, Costa Rica, Cuba, Guatemala, Mexico, Puerto Rico, Panama, and the United States. The pathogen had also been reported as attacking teosinte under the binomial P. sorghi. Only the uredospore and teliospore stages of P. polysora have been reported thus far. The uredospores are 24 to 29 x 27 to 34 μ in size according to Arthur (1934). The teliospores measure 18 to 26 x 29 to 40 μ.

[illegible] has been reported on maize plants from possibly all the maize-growing regions of the World. The pycnial and aecial stages occur on various species of Oxalis and the uredial and telial stages on maize and teosinte. The uredospores measure 23 to 29 x 26 to 32 μ; the teliospores measure 16 to 23 x 29 to 45 μ.

Angiopsora zeae on maize is somewhat similar in appearance in the uredial stage to P. polysora, and it has been confused at least once with P. polysora in Puerto Rico. The uredospores measure 16 to 20 x 22 to 30 μ; the unicellular teliospores are catenulate in sessile chains of usually two spores. This rust fungus, according to Cummins, is known only from Guatemala, Puerto Rico, the Dominican Republic, and Trinidad.

14

The uredospores of the above rust fungi are readily dispersed by wind currents. The germ tubes of the uredospores were observed by Weber (1922) to enter the stomata of a maize leaf either with or without the formation of appressoria.

According to Mains (1931), at least seven physiologic races of P. sorghi have been differentiated. Vallega (1944) reported the existence of at least two physiologic races of P. sorghi in the Llavallol district of the province of Buenos Aires in Argentina.

In most regions the source of inoculum for initial and new infections is the uredospores; however, Weber (1922) concluded that the uredospores did not overwinter in the vicinity of Madison, Wisconsin, in the winter of 1919-1920. The uredospores were found to retain their ability to germinate for 30 to 60 days but declined rapidly thereafter. The time required for initial infection is probably less than

According to Weber (1922), the optimum, maximum, and minimum temperatures for the germination of uredospores of P. sorghi were 17°, 32° and 4°C., respectively. The optimum temperature for infection was 18°C. and the maximum and minimum temperatures somewhere in the vicinity of 32° and 4° C.

Johns and Brown (1941) indicated that P. sorghi was most destructive in regions of high humidity, as in the Gulf States area of the United States. Semeniuk and Wallin (1947) observed that in Guatemala, P. sorghi was the most prevalent and severe disease of maize at elevations ranging from 100 feet o 8,200 feet. They attributed the destructiveness of leaf parasites of maize in Guatemala to the high humidities prevailing in the experimental areas. Wilschire (1953) reported P. polysora as prevalent at elevations of 3,900 feet in Uganda, and Marchionatto (1948) reported P. sorghi as common in the humid part of the cereal region in Argentina.

The control of maize rusts by regulation seems unlikely, since the uredospores can be carried long distances by wind curreents. Likewise, control by cultural methods does n seem very promising, although Johns and Brown (1941) in Louisiana indicated that the date of planting was important. Puccinia sorghi caused serious injury to maize planted around June 15 and July 3 earch year and occasionally damaged the June 1 plantings. Thus it appears that in Louisiana, it is best to plant the maize by June 1 or earlier. It is also quite probable that maize rusts can be controlled by spraying or dusting with the opporpiate chemicals whenever the damage that might ensue warrants such measures.

Wellensiek (1927), Mains (1931), Vallega (1944), and Borlaug (1946) have indicated that varieties and selections of maize exhibit considerable variation in susceptibility to the physiologic races of P. sorghi. Wellensiek worked with two physiologic races of P. sorghi and observed that some selections of maize might be susceptible to one of the races but resistant to the other. He believed that susceptibility in the maize plants was determined by the presence of a relatively large quantity of a certain nutritive substance which acts chemotropically on P. sorghi after pentration, and makes possible an abundant development of mycelium and spores. Thus substance was believed absent or present in only small quantitites in the more resistant selections. Mains indicated that resistance to physiologic Races 1 and 3 of P. sorghi was inherited on a signal-factor pair basis. Valega reported the presence of two physiologic races of P. sorghi in Argentina. Borlaug observed that P. sorghi was relatively common in Mexico, that three species of Oxalis, the alternate host, were rusted heavily each year, and that the native varieties of maise generally more resistant than introduced varieties.

The various species of Sclerospora reported to attack maize plants have been studied most intensively, and have caused their greatest destruction, in the maize-producing regions of the Philippines and Indonesia. These two regions have hot, wet, tropical climates although the climate of Java is supposed to be somewhat drier than that of the Philippines, and Java has a dry winter season. Actually, many of the areas in Java are extremely wet and the rainfall is well distributed throughout the year.

1b

The reports of Sclerospora on maize in the United States havebeen from areas characterized by a humid, microthermal climate with a more or less evenly distributed rainfall and only in years in which there was excessive precipitation during the early growing period of the plants. Sclerospora graminicola and S. macrospora have undoubtedly been present in the maize-producing regions for years, yet they have never become extremely destructive except in definite localized maize plantings where the young plants were subject to flooding for a period of hours or longer. From the data presented it is suggested that species of Sclerospora will be serious pathogens of maize only in regions classified as superhumid or humid, and that their significance will decrease as the humidity of such regions decreases. This implies that the downy mildew disease of maize is important in the Philippines and Java, might be important in the regions of Brazil and China and could be occasionally destructive in localized areas of the United States and Europe. This disease probably will not be of significance in subhumid or arid regions. No information was obtained to indicate that the downy mildew fungus, or fungi has been recorded in Argentina.

The Scleresporas attacking maize produce enormous numbers of sporaggia on plant tissues; these, however, serve to disseminate the fungi only for short distances of a few hundred years, since sporangia are extremely susceptible to desiccation.

Literature Cited

Anon., 1951. Rust epidemic of West African maize. World Crops 3: 220.

Arthur, J. C., 1934. Manual of the rusts in the United States and Canada. 438 pp. Purdue Research Foundation, LaFayette

Borlaug, N. E., 1946. Puccinia sorghi on corn in Mexico. Abstract, 37th annual meeting, Mar. 27-30, 1946. Phytopath. 36: 395.

Campi, M. D., 1939, "Helminthosporium turcicum" Pass. en la Republica Argentina. Lilloa 4: 5-32.

Chester, K. S., 1947. Nature and prevention of plant diseases. 525 pp. The Blakiston Company, Philadelphia.

Christensen, J. J. and E. C. Stakman, 1926. Physiologic specialization and mutation in Ustilago zeae. Phytopath. 15: 979-999.

Cummins, G. B., 1941. Identity and distribution of three rusts of corn. Phytopath. 31: 856-857.

Davis, G. N., 1936. Some of the factors influencing the infection and pathogenicity of Ustilago zeae (Beckm.) Unger on Zea mays L. Iowa Agri. Expt. Sta. Res. Bull. 199.

Dickson, J. G., 1947. Diseases of field crops. 429 pp. McGraw-Hill Book Company, Inc., New York.

Elliott, C. W., 1943. Leaf blight of corn. Phytopath. 33: 407-408.

Garber, R. J. and M. M. Hoover, 1928. The relation of smut infection ot yield in maize. Jour. Am. Soc. Agron. 20: 735-746.

Hoppe, P. E., 1953. Northern corn leaf blight in Wisconsin in 1952. Pl. Dis. Rept. 37: 94.

Immer, F. R. and J. J. Christensen, 1928. Determination of losses due to smut infection in selfed lines of corn. Phytopath. 18: 599-602.

Immer, F. R. and J. J. Christensen, 1931. Further studies on reaction of corn to smut and effect of smut on yield. Phytopath 21: 661-674.

Johns, D. M. and H. B. Brown, 1941. Effect of date of planting on corn yields, insect infestation, and fungous diseases. La. Agri. Expt. Sta. Bull. 327, 28 pp.

Johnson, I. J. and J. J. Christensen, 1935. Relation between number, size, and location of smut infections to reduction in yield of corn. Phytopath. 25: 223-233.

Kernkamp, M. F., 1942. The relative effect of environmental and genetic factors on growth types of Ustilago zeae. Phytopath. 32: 554-567.

Kernkamp, M. F. and W. J. Martin, 1941. The pathogenecity of paired haploid lines of Ustilago zeae versus the pathogenicity of numerous mixed haploids. Phytopath. 31: 1051-1053.

Mains, E. G., 1931. Inheritance of resistance to rust, Puccinia Sorghi, in maize. Jour. Agri. Res. 43: 419-430.

Marchionatto, J. B., 1948. Tratado de fitopatologia. 537 pp. Ediciones Libreria Del Colegio. Buenos Aires.

Melhus, I. E., et al., 1941. Pathology and Mycology of corn. Rep. Ia. Agri. Expt. Sta. 1940-41, Part II. pp. 54-58.

Nattrass, R. M., 1953. Occurrence of Puccinia polysora Underw. In East Africa. Nature 171: 527.

Naumenko, F. E., 1941. [Ecological factors and blister smut of maize]. C. R. Pan-Sov. V. J. Lenin Acad. Agr. Sci., Moscow, 1941, 5, pp. 39-41.

Pepper, B. B. and C. M. Haenseler, 1944. Control of European corn borer and ear smut on sweet corn with dusts and sprays. Cir. 486, N. J. Agri. Expt. Sta. 14pp.

Rhind, D., J. M. Waterston, and F. C. Deighton, 1952. Occurrence of Puccinia polysora Underw. In West Africa. Nature 169: 631.

Semeniuk, G. and J. R. Wallin. 1947. The influence of climate on four leaf parasites Zea mays in Guatemala. Abstract, 38th annual meeting, Dec. 28-30, 1946. Phytopath. 37, 20.

Stakman, E. C. and J. J. Christensen, 1927. Heterothallism in Ustilago zeae. Phytopath. 17: 827-834.

Stakman, E. C., M. F. Kernkamp, T. H. King, and W. J. Martin, 1943. Genetic factors for mutability and mutant characters in Ustilago zeae. Amer. Jour. Bot. 30: 37-48.

Stakman, E. C., J. M. Daly, M. L. Gattani, and I. Wahl. 1948. Variation induced by uranium nitrate in corn smut and the cultivated mushroom. Science 108: 554-555.

Stringfield, G. H. and D. H. Bowman, 1942. Breeding corn hybrids for smut resistance. Jour. Amer. Soc. Agron. 34: 486-494.

Ullstrup, A. J., 1941a, Inheritance of susceptibility to infection by Helminthosporium maydis Race 1 in maize. Jour. Agri. Res. 63: 331-334.

Ullstrup, A. J., 1941b. Two physiologic races of Helminthosporium maydis in the corn belt. Phytopath. 31: 508-521.

Ullstrup, A. J., 1943. Diseases of dent corn in the United States. Circ. 674, U.S.D.A., 34 pp.

Ullstrup, A. J., 1944. Further studies on a species of Helminthosporium parasitizing corn. Phytopath. 34: 214-222.

Vallega, J., 1944. Observaciones preliminares sobre especializacion fisiologica de Puccinia sorghi, en Argentina. An. Inst. Fitotec. S. Catalina, 1942, 4: 14-18.

Walker, E. A. and J. W. Magruder, 1943. Maryland field corn leaf blight disease survey - 1942. Pl. Dis. Rept. 27: 126-135.

Walter, J. M., 1934. The mode of entrance of Ustilago zeae into corn. Phytopath. 24: 1012-1020.

Weber, G. F., 1922. Studies in corn rust. Phytopath. 12: 89-97.

Wellensiek, S. J., 1927. The nature of resistance in Zea mays L. to Puccinia sorghi Schw. Phytopath. 17: 815-825

Wernham, C. C., 1946. Three hitherto unreported diseases of corn in Pennsylvania. Pl. Dis. Rept. 30: 26-28.

[illegible], S. P., 1953. Uganda: Outbreaks of New Diseases. FAO Pl. Prot. Bull. 1, p. 62.

[illegible], H. and R. Salzmann, 1947. Pflanzenschutz. etc. Bericht uber die Tatigkeit der Eidg. Landwirtschaftlichen Versuchsanstalt. Zurich-Oerlikon fur die Jahre 1942-1946. Annu. Agr. Suisse. 48: 234-224.